DISCUSSION POINTS

OSHA'S PROPOSED RULE ON WORKPLACE IAQ

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WHAT IS ENVIRONMENTAL TOBACCO SMOKE?

Claims: "ETS contains substances which are known to cause cancer in humans or animals and many of which are strong irritants."

Response:

- ETS is a highly diluted constituent of an indoor environment that is comprised of aged, diluted sidestream smoke and exhaled mainstream smoke:

> Mainstream smoke is the tobacco smoke that is drawn through the butt end of a cigarette during active smoking; this is the tobacco smoke that the active smoker inhales into his or her lungs. Sidestream smoke is the tobacco smoke that is released in the surrounding environment of the burning cigarette from its smoldering tip between active puffs. Many publications have treated sidestream smoke and ETS as if they were one and the same, but the sidestream smoke and ETS are clearly not the same thing. Sidestream smoke and ETS have different physical properties and they have different chemical properties. Environmental tobacco smoke is usually defined as a combination of highly diluted sidestream smoke plus a smaller amount of that residual mainstream smoke that is exhaled and not retained by the active smoker. What really is ETS? In comparison to mainstream smoke and sidestream smoke, ETS is so highly diluted that it is not even appropriate to call it smoke, in the conventional sense. Indeed, the term "environmental tobacco smoke" is a misnomer. Huber, et al., "Passive Smoking:

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How Great Hazard," Consumer's Research, pp. 10, 11-12 (July 1991).

- Environmental tobacco smoke (ETS) is an aged and dilute mixture of sidestream smoke (SS; the smoke from the burning end of the cigarette) and exhaled mainstream smoke (MS; the smoke to which the smoker is exposed). ETS differs chemically and physically from both MS and SS. ETS is a dynamic, ever-changing mixture which, as it ages and dissipates, undergoes chemical reactions and physical change. There is no single definable, reproducibly characterizable entity known as ETS,[1] to be found in the workplace, in bars or in restaurants.

- Dissipative forces such as air currents and attraction to surfaces influence SS and exhaled MS.[1] Studies indicate that constituents in ETS are hundreds to thousands of times more dilute than either SS or MS. Often, workplace concentrations of ETS constituents fall below detection limits of current scientific measurement devices.[1]

- As ETS ages, a number of physico-chemical changes take place. Matter evaporates from SS particles as they age to ETS. During the aging process, ETS particles coagulate and increase in size. Chemical compounds partition between the gas and particle phase of the smoke. (For example, nicotine is found in the particle phase of MS; in fresh SS, most of the nicotine

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is in the gas phase.) Decay patterns for constituents of ETS vary over time and are dependent upon physical conditions in the environment.

- ETS is not equivalent to either MS or SS. Many studies and reviews employ sidestream/mainstream smoke comparisons, ostensibly to demonstrate the kind and quantity of constituents involved in exposure to ETS. But such comparisons are deceptive and misleading. As two tobacco smoke chemists reported:[1]

> Although ETS originates from sidestream and exhaled mainstream smoke, the great dilution and other changes which these smoke streams undergo as they form ETS make their properties significantly different from those of ETS. Thus, the sidestream/mainstream ratios quoted in Table 1 can be misleading if used out of context. The important question is not the ratio of sidestream/mainstream but rather what is the concentration of the constituent in the indoor environment and how does it compare to levels from sources other than ETS. Studies based solely on observations of fresh sidestream, or highly and unrealistically concentrated ETS, should take into account the possible differences between these smokes and ETS found in real-life situations.

- Even the 1986 Report of the Surgeon General on ETS conceded:

> Comparison of the relative concentrations of various components of SS and MS smoke provides limited insights concerning the toxicological potential of ETS in comparison with active smoking.

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As described above, SS characteristics, as measured in a chamber, do not represent those of ETS, as inhaled by the non-smoker under non-experimental conditions.[2]

- Similarly, the 1986 NAS/NRC Report on ETS concluded:

  Because the physicochemical nature of ETS, MS, and SS differ, the extrapolation of health effects from studies of MS or of active smokers to nonsmokers exposed to ETS may not be appropriate.[3]

- All three publicly available animal inhalation studies have reported no statistically significant increased incidence of tumors in animals exposed to ETS compared with control animals.[4-6]

- Moreover, none of the lifetime, "whole smoke" exposure studies on animals, originally designed to assess possible biological effects from mainstream smoke exposures, have reported the induction of lung carcinomas via inhalation.

- Recent reviews of the literature on suspected pulmonary carcinogens have concluded that none of the individual constituents in sidestream smoke that is classified as potentially carcinogenic has been found to induce pulmonary cancer via inhalation in experimental animals.

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- The "carcinogenic agents" supposedly identified in tobacco smoke either are not suspected pulmonary carcinogens, or have not been unequivocally demonstrated as tumorigenic to human tissue or to the lung tissue of experimental animals.[7-10]

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ETS EXPOSURE AND LUNG CANCER IN NONSMOKERS

Claims: "ETS has been classified by the U.S. Environmental Protection Agency (EPA) as a known cause of lung cancer in humans (Group A carcinogen)."

"Exposure to ETS is estimated by EPA to cause approximately 3,000 lung cancer deaths in nonsmokers each year."

Response:

- The claim of 3,000 deaths purportedly due to ETS exposure is derived from a risk assessment on ETS conducted by the U.S. Environmental Protection Agency. The risk assessment was based upon a statistical review, called "meta-analysis," of epidemiologic studies of nonsmoking women married to smokers.

- The meta-analysis conducted by EPA used 11 spousal smoking studies conducted in the United States.[1-11] Not one of these studies originally reported a statistically significant overall risk estimate.

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- Indeed, even when the EPA lowered the statistical confidence limits reported for the U.S. studies from the standard 95% to 90%, all but one of the 11 studies individually failed to achieve statistical significance.[5]

- Nevertheless, EPA used these studies to arrive at the conclusion that ETS exposure is associated with an increased risk of lung cancer.

- The EPA failed to include in its meta-analysis relevant data from two recent, major U.S. studies on ETS.[12,13] These two studies appeared in the scientific literature months prior to the release of the Risk Assessment.

- The inclusion of both the studies in the meta-analysis should negate the classification of ETS as a Group A carcinogen, in that a cumulative risk estimate for the U.S. studies would not be statistically significant, even at the 90% confidence interval.[14] The Agency should have estimated no statistically significant increased risk for lung cancer, at either a 95% or a 90% confidence interval.

- A critical flaw of the EPA's meta-analysis is that it did not aggregate similar data. For example, all of the studies employed different questionnaires and different assessments of

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past exposure; five of the studies failed to match cases with controls; and the criteria for control selection were different in each study.[15]

- EPA has not addressed the issue of the aggregation of dissimilar data in its meta-analysis. Moreover, even the "common thread" identified by the EPA as justification for meta-analysis, spousal smoking, is defined differently across studies.

- Furthermore, EPA reviewed a total of 30 epidemiologic studies in the risk assessment.[1-11,16-34] Of these, 24 reported no statistically significant overall risk estimate, meaning that these data are compatible with the hypothesis that there is no overall association between spousal smoking and lung cancer.

- Even positive point estimates (greater than 1.00) that do not achieve statistical significance are compatible with the null hypothesis of "no association" between spousal smoking and lung cancer. Reported overall point estimates in six non-U.S. studies are statistically significant, but the EPA did not ascertain whether or not these purported associations are due to some feature that is coincidental to "marriage to a smoker" (e.g., common diet, occupational exposures, socio-economic status and other lifestyle characteristics).

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- During the course of the development of the risk assessment, numerous critical scientific comments were provided to the Agency. For instance, more than 100 public comments were submitted on the first public draft of the risk assessment, in 1990. An EPA consultant involved in the risk assessment characterized the majority of the comments as "quite lengthy, detailed, and highly critical."

- Many of the points raised in the public comments in 1990, and in additional public comments provided to EPA in 1991, were not adequately addressed by the Agency before the risk assessment was finalized. For instance, a meta-analysis incorporating the two new United States studies was submitted to the Executive Committee of the EPA's Science Advisory Board; it yielded a summary risk estimate that was not statistically significant.[14] Nevertheless, the risk assessment was finalized without taking into account these new data.

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ETS EXPOSURE AND CLAIMS OF IRRITATION AND ANNOYANCE

Claims: "Exposure to ETS in the workplace causes irritation of the eye, nose, and throat."

Responses:

- Persons may report "discomfort" or "annoyance" that they attribute to ETS exposure. However, subjective perceptions are difficult to evaluate.

- The existence of allergens in tobacco smoke has not been established conclusively.[1-3] Claims of annoyance and emotional responses should not be confused with genuine allergic reactions.

- A vast range of substances contribute to workplace indoor air quality. Singling out ETS for particular attention, as OSHA has done in its proposed rule, overlooks these other substances. Personal perceptions of comfort may be related to air temperature, relative humidity, lighting, and/or the presence of biological and chemical substances in the air.

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ETS EXPOSURE AND ADULT RESPIRATORY CONDITIONS

Claims: "ETS exposure in the workplace can also irritate the lungs, leading to coughing, excess phlegm, chest discomfort, and reduced lung function."

Responses:

- Even the authors of the EPA risk assessment on ETS concede that the studies on ETS and adult respiratory health are "inconsistent and sometimes contradictory."

- The U.S. Surgeon General, in the 1986 report on ETS, concluded that the magnitude of the reported lung function decrements in nonsmoking adults exposed to ETS were "quite small, even at moderate to high exposure levels" and that "it is unlikely that this change in airflow, per se, results in symptoms."[1]

- Regarding cough, phlegm and wheezing in adult nonsmokers, the Surgeon General reported that "few studies have addressed the relationship of chronic respiratory symptoms in nonsmoking adults with environmental tobacco smoke exposure." The Surgeon General reported on two studies (Schilling 1977; and Schenker 1982). Neither study reported an association between

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spousal smoking and an increased incidance of respiratory symptoms in nonsmoking women.

- Several literature reviews on ETS and adult respiratory health (not addressed in the EPA risk assessment) have contradicted the claim that ETS exposure is associated with respiratory symptoms in nonsmoking adults. For example, a review by Laurent, et al., (1989) suggested that the purported long-term health effects from exposure to ETS are difficult to demonstrate in healthy adults and that the results of the epidemiological studies are "sometimes conflicting and often open to question." In a 1990 review, Witorsch noted that, in addition to "all of the problems" with the studies, "these results are too variable to permit any conclusion of association."

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ETS EXPOSURE AND CARDIOVASCULAR DISEASE

Claims: "Exposure to ETS in the workplace may affect the cardiovascular system, and some studies have linked exposure to secondhand smoke with the onset of chest pain."

Responses:

- Although there are 14 epidemiological studies on a possible association between ETS and heart disease, in most of these, the data were not reported to be statistically significant.

- All of the studies suffer from a variety of serious methodological weaknesses including small sample sizes, misclassification of smoking status of study participants, inadequate assessment of ETS exposure, failure to control adequately for potential confounding variables and failure to confirm causes of death.

- These weaknesses in the data were emphasized in the 1986 Surgeon General's Report which judged that "no firm conclusion" could be made regarding a possible relationship between ETS and heart disease.[1] A 1986 report from the National Academy of Sciences (NAS) reached a similar

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conclusion.2

- A review of the literature on ETS and heart disease was published in 1991 by the former director of toxicology of a major European research laboratory. In it, he described the scientific data relating to a possible heart disease risk in nonsmokers exposed to ETS as "not very convincing."[3]

- The claim about the onset of chest pain (angina) is largely based on a 1978 study involving 10 heart patients which reported that exposure to tobacco smoke could hasten the onset of heart pain during exercise.[4] However, the credibility of this report, as well as other research by this investigator, has been widely challenged.[5]

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INDOOR AIR QUALITY IN SCHOOLS/DAYCARE CENTERS ILLUSTRATES THE SERIOUSNESS OF IAQ PROBLEMS AND DEMONSTRATES THE NEED FOR COMPREHENSIVE APPROACHES

- Because most children in the United States spend a large amount of time in schools and daycare centers, and because environmental factors have been shown to be critical in a child's ability to concentrate and learn, indoor air quality in these facilities has become an increasingly important issue. The seriousness of these IAQ problems (where smoking is not involved) underscores the need for OSHA to pursue all aspects of indoor air quality.

- The U.S. EPA's 'School Evaluation Program' (SEP) began in 1989 to provide information for diagnostic and mitigation strategies in schools with elevated radon levels. Twenty-six schools across the U.S. have been evaluated. In 1991, investigators reported that "diagnostic data showed a strong trend that radon was not the only indoor air pollutant in these schools." The researchers also reported that, in general, ventilation was "inadequate" and below the current ASHRAE Standard 62-1989 guidelines. The authors of the report stated the following:

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During the process of conducting these evaluations, many schools were found to have disabled or malfunctioning outdoor air supply. Although these schools had been chosen for evaluation because they had been identified as having elevated radon levels, results of these evaluations indicate the need for a new direction in large building radon abatement -- a holistic (buildings systems) approach that considers total indoor air quality, comfort, cost and energy issues.[1]

- Poor indoor air quality in schools has been called "the asbestos of the '90s."[2] The World Health Organization estimates that 30% of the nation's schools are suffering from the "sick school syndrome."[2] In fact, according to some, the figure may be even higher for schools.

- A recent report from the New York State United Teachers Union suggests that dozens of New York City's schools are poorly ventilated and may present health hazards to children.[4] The union reported evidence of poor ventilation and chemical contamination in approximately one in 10 schools they investigated statewide.[4] The union reported, however, that the actual number of sick schools may be as high as one in two.[5] In Hartford, Connecticut, it has been reported that eight of 30 school buildings were found to have ventilation problems.[6]

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- Similarly, the authors of a 1990 study of six primary schools reported that "the mean concentration of carbon dioxide was above the recommended value" in all of the schools, indicating poor outdoor air supply.[7] The authors concluded that "sick building syndrome" in schools is a problem of multifactorial origin.

- Many scientists appear to believe that schools are especially susceptible to "sick building syndrome," due to the large number of people per room.[2, 8] Budget restraints are also often cited as an important factor in "sick school syndrome."[5, 2, 9] One author reported that a lack of fresh air, improperly applied pesticides, and offgassing from furnishings and carpets are also to blame. Thomas Hobart, the President of the New York State United Teachers Union, reported:

> In many schools, budget cuts have led to poor maintenance, inadequate cleaning and measures that were intended to tighten buildings for energy savings but have resulted in poor air quality.

- A review of the literature on sick schools reveals that symptoms and complaints include rashes, headaches, respiratory problems, allergic reactions, multiple chemical sensitivity, fainting, eye and throat irritation, an inability to

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concentrate, stomach aches, fatigue and nausea.6, 10 As one author stated:

> As most youths in the industrialized world spend a decade or more in schools, impairment of health due to poor quality of indoor air in schools may have implications for the health state of a large proportion of the population.7

- An informal review of media reports that discuss reported incidents of "sick" schools and daycare centers indicates that problems associated with poor IAQ are usually in large part due to overcrowding, uncleanliness or poor building/HVAC design. In fact, smoking is prohibited in most of these schools. This review did not identify a single instance where ETS exposure was blamed for poor IAQ in schools or daycare centers.

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THE PROPOSED OSHA RULE DOES NOT ADDRESS TUBERCULOSIS AND INDOOR AIR QUALITY

- Tuberculosis (TB) is a mycobacterial disease that is important as a cause of disability and death in many parts of the world. Initial infection with TB usually goes unnoticed; tuberculin sensitivity appears within a few weeks. For many years, the United States has shown downward trends of TB. However, new multi-drug resistant strains of TB and the growing population of HIV-infected individuals have increased the incidence of TB once more. The resurgence of TB infections has health officials once again analyzing environmental conditions as possible control mechanisms.

- In the United States, 15 million people are infected with tuberculosis.[1]

- Mycobacteria may only be transmitted by water droplets inhaled deeply into the lungs. Direct invasion of TB through mucous membranes or breaks in the skin may occur, but is extremely rare. The degree of communicability depends on the number of bacilli discharged, the virulence of the bacilli and opportunities for their aerosolization by coughing, sneezing, talking or singing. Early studies of TB suggested a direct relationship between shared air supply and infection: in

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addition to individual susceptibility, exposure to an infected air stream was a factor in the spread of TB.[2]

- Dr. Edward Nardell, a TB Control Officer for the Massachusetts Department of Public Health, has analyzed the potential impact of outdoor air supply rates on infection rates of TB. Nardell investigated a case where 27 out of 67 building occupants (40%) were infected from the spread of the disease from a single individual who had contracted the disease. According to Nardell's data, reducing the outdoor air rate to 5 cfm/person would have doubled the rate of infection, whereas increasing it to 25 cfm/person would have reduced it by a third. Raising the ventilation to 35 cfm/person would reduce the rate of infection by half. Nardell concluded that increasing ventilation reduces the chance of infection, but does not eliminate it. He suggests that increasing the outside ventilation rate could have a significant impact in reducing TB infection rates in office buildings.[3]

- Dr. Harriet Burge reported that infectious illnesses that are spread by aerosols can spread more rapidly in poorly ventilated spaces.[3] She writes:

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> Airborne transmission usually occurs only when an infected subject is coughing, sneezing, or otherwise actively shedding fresh organisms into air close to susceptible individuals. In addition, when shedding occurs in poorly ventilated environments, levels can build to the point where susceptible subjects throughout an occupied space become infected. [authors emphasis]

- In addition to increased ventilation, Dr. Nardell suggests that ultraviolet light may be effective in disinfecting the indoor air.[4] He writes:

> Increased outdoor air ventilation is not the only way to disinfect building air. The ability of ultraviolet (UV) irradiation of the 254 nm wavelength to render indoor air noninfectious has been demonstrated in experimental exposure chambers, in experimental rooms, and, to a lesser extent, by field trials. Species of microorganisms vary in their susceptibility to UV irradiation, but many bacteria, including tubercle bacilli, and some airborne viruses are killed with levels of UV irradiation that are both practical and safe for certain applications.

- Dr. Nardell did not suggest that office buildings be equipped with UV fixtures for air disinfection. However, if a TB problem exists in a particular building, he feels that UV fixtures may supplement the protection afforded by outdoor ventilation.

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